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Isolated hepatosplenic abscess from cat scratch disease in a patient with HIV

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ABSTRACT

Bartonella henselae infection, or cat scratch disease, typically is a self-limiting disease presenting as lymphadenopathy and fever after a bite or scratch from a cat. The most commonly reported presentation in immunocompromised patients includes bacillary angiomatosis and peliosis hepatitis, which resemble Kaposi's sarcoma. Isolated hepatosplenic abscess without diffuse lymphadenopathy or vasoproliferative disease is seldom reported in adult immunocompromised patients. Although several advances have been made in identifying the organism and antibodies with serological tests, biopsy, and polymerase chain reaction, there is little information about treatment. We report a case of an isolated hepatosplenic abscess without lymphadenopathy or vasoproliferative disease caused by *B. henselae* in an adult immunocompromised patient with HIV.

KEYWORDS *Bartonella henselae*; cat scratch disease; hepatosplenic abscess; HIV; immunocompromised

Bartonella species are facultative gram-negative bacteria known to cause disease in humans due to cat bites or scratch.[1,2] *Bartonella henselae* is the most frequently identified agent for cat scratch disease (CSD), with domestic cats being the primary reservoir.[2] CSD can present with disseminated disease in immunocompromised patients. We report a case of a man with human immunodeficiency virus (HIV) infection with hepatosplenic abscess from *B. henselae* without signs of diffuse vascular involvement or lymphadenopathy.

CASE PRESENTATION

A 48-year-old man came to the hospital for evaluation of fever, chills, weakness, dyspnea, dysuria, and chest and abdominal pain. The symptoms started after initiation of HIV treatment 1 week earlier. The HIV infection had been diagnosed 13 years earlier. He reported untreated hepatitis C infection and a history of intravenous drug use. His temperature was 100.9°F. There was abdominal tenderness and track marks on his left arm. The leukocyte count was 4700/U; aspartate transferase, 146 U/L; alanine aminotransferase, 97 U/L; alkaline phosphatase, 104 U/L; and erythrocyte sedimentation rate, 68 mm/h. Computed tomography angiography of the chest revealed splenomegaly (up to 17.5 cm). He was started on broad-spectrum antibiotics with intravenous vancomycin and piperacillin-tazobactam. His CD4 count was 504 (36%), and blood cultures were negative for >48 hours. The echocardiogram was normal. Magnetic resonance imaging (MRI) of the abdomen revealed hepatosplenomegaly with multiple liver microabscesses measuring up to 5 mm *(Figures 1a, 1b)*. Antibiotics were deescalated to ceftriaxone. Ultrasound-guided biopsy of the liver abscess revealed chronic hepatitis and steatosis. Cultures of the abscess grew *Enterococcus faecalis,* and antibiotics were changed to amoxicillin-clavulanic acid.

Due to the patient's failure to improve clinically and interval development of an enlarging hepatosplenic abscess *(Figures 1c, 1d)*. initial culture results were considered a contaminant. Extensive tests were done, including a serum beta-D-glucan test for candidiasis; serologic tests for IgG and IgM antibodies against *Bartonella* spp., *Francisella tularensis, Brucella* spp., and *Coxiella burnetii*; polymerase chain reaction test for cytomegalovirus; serum cryptococcal antigen; urine *Histoplasma* antigen; and QuantiFERON Gold test for

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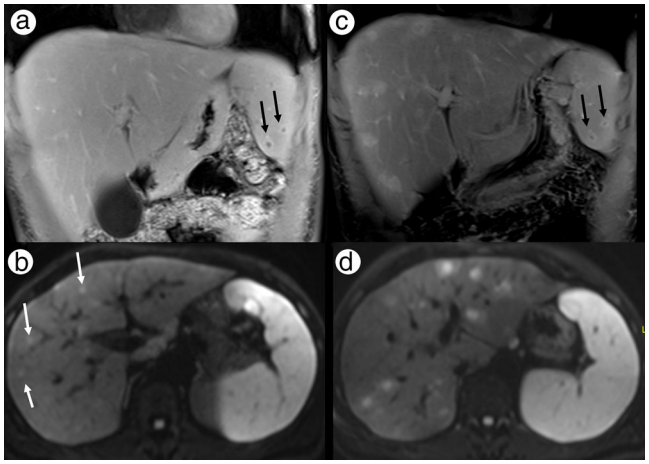

Figure 1. In the initial MRI, the **(a)** postcontrast coronal image showed multiple tiny rim-enhancing lesions in the spleen (black arrows), and the **(b)** diffusion-weighted image showed multiple tiny foci of diffusion restrictions in the liver (white arrows), compatible with micro abscesses. The liver lesions were not visualized in the contrast images. The MRI done 2 weeks later showed progression in the number and size of liver lesions. Lesions were visible in both **(c)** postcontrast and **(b)** diffusion-weighted images. The splenic lesions were stable (black arrows).

tuberculosis. Results showed positive IgG titers at 1:512 for *B. henselae*. A history of exposure to cats was elicited. Repeat biopsy of the abscess and a polymerase chain reaction test for *B. henselae* returned positive. The patient showed an excellent clinical response to 2 weeks of intravenous gentamicin and 4 weeks of oral doxycycline before discharge to home.

DISCUSSION

B. henselae is a small pleomorphic, fastidious, facultative gram-negative bacillus that was first observed from a lymph node of a patient infected with CSD in 1983.[1,3] The average annual incidence of CSD is reported to be 0.7 to 0.8 per 100,000 population.[4,5] CSD is frequently seen in children aged <14 years, followed by adults. The organism's affinity to the vascular endothelium and its effects on vascular endothelial growth factor are thought to cause its intraerythrocytic proliferation.[2,6] Typical CSD frequently presents as an erythematous papule followed by isolated regional lymphadenopathy with or without fever. It is considered a self-limiting illness rarely requiring treatment, including antibiotics or lymph node drainage.[2] Immunocompromised patients frequently have atypical presentations such as retinitis, Parinaud oculoglandular syndrome, hepatosplenic abscess, endocarditis, osteomyelitis, and vasoproliferative manifestations like bacillary angiomatosis and bacillary peliosis.[2,5] Diagnosis of atypical CSD can be challenging in the absence of relevant clinical history. Blood cultures and tissue cultures have prolonged incubation periods.[2] The polymerase chain reaction and serological tests are used in diagnosis.[2,7–9]

Treatment is currently dictated by published cases based on physician discretion and in vitro studies.[10,11] In atypical cases, a combination regimen such as doxycycline with rifampin or gentamicin or erythromycin for durations ranging from 2 weeks to 2 months has been reported.[10–13] With the high prevalence of domestication of cats in the current era, *B. henselae* can be an emerging infection. The use of appropriate antibiotics for treatment can have an excellent clinical response in immunocompromised patients, as demonstrated in our case.

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